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ABSTRACT

Proposed is a study to evaluate the auditory systems of learning disabled (LD) students with a new audiological, diagnostic, stimulus apparatus which is capable of objectively measuring the interaction of the binaural aspects of hearing. The author points out problems with LD definitions that exclude neurological disorders. The detection of central hearing involvement as well as the expansion of MAID (minimal auditory intensity differential) stimulus for proposed control testing are briefly discussed; and data from a pilot study are presented. The bulk of the document consists of tables and charts explaining the use of the MAID stimulus. (SBH)

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A STUDY OF THE ROLE OF CENTRAL AUDITORY PROCESSING IN LEARNING DISABILITIES

BY

HUGH MURRAY, B.A.

A Prospectus Submitted to the

Department of Speech

New Mexico State University

Las Cruces, New Mexico

May, 1976

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Introduction

It is generally agreed that many LD problems are caused by "auditory" deficiencies. The concept of "auditory" may vary, however. For example, the National Advisory Committee for the Handicapped (1971) set forth the following definition of specific LD:

> Children with specific (special) Learning Disabilities exhibit a disorder in one or more of the basic psychological processes involved in understanding or in using spoken or written language. These may be manifested in disorders of listening, thinking, talking, reading, writing, spelling or arithmetic. They include conditions which have been referred to as perceptual handicaps, brain injury, minimal brain dysfunction, dyslexia, developmental aphasia, etc.

In this definition, "listening" is included but "hearing" is excluded. One can only assume from definitions that writers consider these mutually exclusive terms, or perhaps view "listening" as psychological and "hearing" as physiological. Perhaps we are caught in the semantic game of word meaning, or perhaps in the general problem of the semantic aspect of labels expressing actuality. Kirk (1971) further hazes the problem for this writer in his summary statement:

> The concept of learning disability demands a relatively new educational approach which involves handicapped children with specific disorders in perceiving, thinking, listening, talking, reading, writing, spelling, arithmetic and related disabilities primarily in the communicative process. Although there may be an overlap among these disabilities and other handicaps, these disorders are discrete as related to the traditional categories of such handicapping conditions as mental retardation, emotionally disturbed, crippling conditions, deafness, blindness and speech defects, all of which are included in special education programs.

Again the dichotomy of perceiving-listening versus perceiving-deafness (one must assume that Kirk allows all degrees of hearing loss to fall under deafness) is spelled out specifically. It is assumed at this point that most experts in LD express the opinion that a child can have a learning disability without a specific related neurological disorder and that by definition "without" is the rule. Even more firmly stated is the Department of Health, Education, and Welfare's definition (1974) which states:

> . . . these learning disorders include perceptual handicaps, brain injury, minimal brain dysfunction, dyslexia, developmental aphasia, etc. but do not include visual, hearing, motor handicaps, mental

> retardation, emotional disturbance or environmental deprivation.

Many audiologists have noted, clinically, a particular drop at 8000 Hz unilaterally in LD kids. However, no published report can be found relating to this observation. The only published mention of hearing as such has to do with the quality of discrimination, since if a hearing loss is present, by definition, an LD situation does not exist. The writer of the prospectus must then ask these questions: (1) If the observations are correct (regarding the audiometric picture), are the children with the particular audiogram LD's or are they hearing impaired? (2) If they are not LD's, then shouldn't we study the labeling procedure to prevent mis-labeling?

An excellent article by Denckla (1974) suggests that "splitting" and "lumping" should be avoided to prevent LD's from falling into the same trap as those suffering Huntington's Chorea in earlier times. That hereditary, degenerative disease was considered functional until systematic investigation found "hard signs" that removed the disorder from the psychiatric realm. Denckla suggests we often let theory get in the way of fact and that theory holders often tend to evaluate the contribution of the specialist (in the case neurology) by standards appropriate at the time of the theory eruption which does not allow for growth or new development. Further, Denckla divides her LD syndromes into three groups, of which she says:

> Finally, I submit that not only syndrome 3 (Dyscontrol) constitutes "neurological impairment" but so do syndromes 1 and 2 and the other 70% of my patients who present other combinations, permutations, and variations of signs. By splitting clinically into symptom complexes or syndromes, we may begin to find out causes (which are due to brain injury, to hereditary deficiencies, to late maturational patterns or to metabolic diseases), and design rational curative and preventive programs, rather than leap to premature panaceas, pseudo-explanations and so-called "beliefs."

Clearly Denckla feels that LD has a neurological base. She also very strongly states a case for additional research.

Those who advocate treating the symptom and ignoring the underlying cause fail to recognize the importance of perceptual substitution and related successive approximation techniques in dealing with known brain damage syndromes such as childhood aphasia. If indeed many LD problems result from minor specific damage at a major synthesizing site along a given neural pathway, then no amount of information (general or specific, concrete or abstract) will be assimilated and used through that site. This should particularly hold true in auditory areas since the auditory

system is the first input mode used to build the reversed pyramid of language. An obvious example of this is the absence of abstract language (verbal or graphic) in deaf children.

The purpose of this proposed study is not to debate the merits of existing programs nor to attempt to measure their worth. Rather, its intent is to evaluate the auditory system with a new audiological, diagnostic, stimulus apparatus which is capable of objectively measuring the interaction of the binaural aspects of hearing. The two aspects to be measured are the binaural summation and the mid-plane phase balancing. Both of these are central tasks requiring normal auditory pathways from the junction of the eighth nerve at the cochlear nucleus. A patented prototype of the MAID now exists; a rough draft of the dual channel version is now being used to collect data on normal children and adults.

Detection of Central Hearing Involvement

Diagnostic effort beyond the eighth cranial nerve is limited to two general areas: (1) speech under varying parameter changes and (2) localization.

The speech tests consist mainly of filtered speech (Jerger, 1960; 1964), accelerated speech and distorted speech (Bocca and Calearo, 1963), and competing speech tests (Bocca and Calearo, 1963; Jerger, 1960; Quiros, 1964). The basis of the speech tests is the supposition that neural integration of a verbal stimuli will occur when the signal has been altered in such a way that normal peripheral hearing is required to adequately interrupt meaning. Perhaps the most widely researched approach using speech is the "filtered speech test." However, Matkin and Olsen (1971) point out that filtered speech has "inherent limitations" of a widely diverse nature. Most approaches using speech are labeled as "promising" with little data to support a stronger statement. Research in the area of speech stimulation has been in progress since 1954 (Bocca and Calearo). An excellent summary of diagnostic speech research efforts written by the two main contributors, Bocca and Calearo, appears in Modern Developments in Audiology (Jerger, 1963).

Localization tests have employed pure tone as well as clicks and complex noises (Jerger, 1960a; Jerger, 1960b; Davis and Goodman, 1966; Sanchez, et al., 1957; Sanchez and Forster, 1958). The major finding in the localization studies is the "decruitment" phenomenon which is reflected by the intensity imbalance occurring across the two test ears to cause perception of a single stimulus at the middle of the head. Specifically, as explained by the researchers, a cortical lesion will be reflected by an increase in loudness in the "good" ear in order to equal the loudness in the "bad" ear. Again, Matkin and Olsen (1971) point out that ". . . it is quite apparent audiology has yet to develop definite tests for the detection of central auditory lesions."

Expansion of MAID For Central Hearing Disorder

Proposed Control Testing Procedure and Apparatus

Neural integration appears to be the major physiological feature present in current central audiological theory and practice. Electrophysiological mapping has shown that many of the assumptions concerning neural integration have a physiological base. Therefore, it would appear that the "clinical study of the central facilitation or inhibition of sensory impulses" as suggested by Bocca and Calearo (1963) is the logical route to follow.

The MAID stimulus is well suited to the sensory impulse approach since it appears to be well established that a complex and continuous stimulus is required for central auditory testing. In peripheral testing, one ear at a time is generally tested; in central testing, the interaction

of both ears is the focus. The following discussion and mathematical model is intended to demonstrate how the MAID is suited to central auditory lesion detection.

The existence of objective audiometry is doubtful; diagnosis is dependent upon the subjective analysis of stimuli by the patient and the stimulus-response relationship evaluated by the examiner. Thus, for the purposes of this model, consideration is upon the in-out relations of the whole model as illustrated in the transfer function shown in Figure 1.

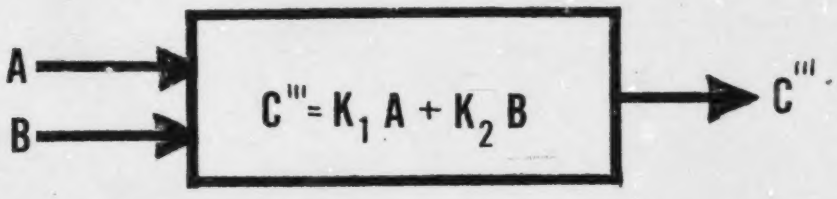

Figure 1

The MAID stimuli at A and B can be represented by an amplitude modulated (AM) sine wave where the modulating signal is a symmetrical square wave with the positive and negative going edges of the square wave coinciding with the zero crossing of the sine wave. The following assumptions will be made for the signals applied at A and B:

a. The peak amplitude and frequency of the modulated sine wave are equal.

b. The frequency of the modulating square wave for A and B are equal and of a much lower frequency than the sine wave.

c. The modulating signal at A will be used as the reference signal for the phrase-or-time difference in the modulating signal for B.

Therefore:

Defining the modulating square wave for A as:

$M_A(t) = M_1$ for $0<t<T/2$

$= M_1$ for $T/2<t<T$

Where $M_A(t) = M_A(t + NT)$, $N = 1, 2, \ldots$

And defining the modulating square wave for B as:

$M_B(t) = -M_2$ for $0<t<b$

$= M_2$ for $b<t<b + T/2$

$= M_2$ for $b + T/2<t<T-b$

Where $M_B(t) = M_B(t + NT)$, $N = 1, 2, \ldots$

Thus:

$$(1)C'''(t) = [K_1(1 + M_A[t])] \sin (W_c t) + [K_2(1 + M_B[t])] \sin (W_c t)$$

$$= A_{12}[1 + M_{AB}(t)] \sin (W_c t)$$

Thus, the output $C'''(t)$ is an amplitude modulated sine wave equal in frequency to A and B and:

1) Overall magnitude factor $A_{12} = K_k + K_2$

2) Modulating signal $M_{AB}(t) = (K_1 M_A[t] + K_2 M_B[t])/(K_1 + K_2)$

Next follows the definition of the input signal at A plus two reference quantities at the input A.

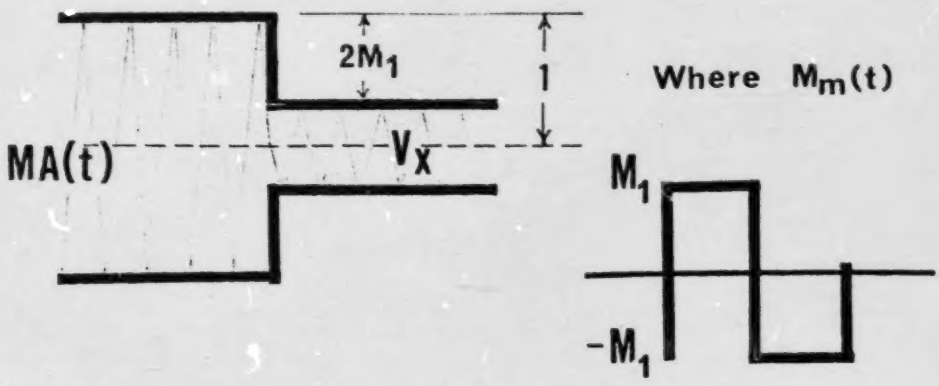

Figure 2

Thus, $A(t) = [(1 - M_1) + M_M(t)] \sin (W_c t)$

Reference intensity at A = RIA = $20 \log_{10}$[Peak of A(t)]

Change in intensity at A = ΔIA = $20 \log_{10}$[Peak of A(t)]/[MIN of A(t)]

Case I: $[A(t) = B(t)]$

$$C'''(t) = (K_1 + K_2)\,[(1 - M_1) + M_M(t)]\sin(W_c t)$$

Thus:

$RIC''' = 20 \log_{10}(K_1 + K_2)$ = Reference intensity at C'''

$\Delta IC''' - 20 \log_{10}[1/1 - 2M_1] = \Delta IA$ = Change in intensity at C'''

Thus:

1) overall increase in intensity at $20 \log_{10}(K_1 + K_2)$ over RIA at the output C'''

2) $\Delta IC''' = \Delta IA$

Case II: $[M_B(t) = -M_A(t)]$

$$C''' = (K_1 + K_2)\left[(1 - M_1) + \frac{K_1 - K_2}{K_1 + K_2} M_A(t)\right]\sin(W_c t)$$

Note that $\dfrac{K_1 - K_2}{K_1 + K_2}$ can be negative if $K_2 > K_1$ and positive if $K_1 > K_2$.

Let $K = ABS[(K_1 - K_2)/(K_1 + K_2)]$

Thus $C'''(t) = (K_1 + K_2)[(1 - M_1) + K(M_A[t])]\sin(W_c t)$

Note that if $K>0$, the peak of $C'''(t)$ is in phase with $A(t)$.

If $K>0$, the peak of $C'''(t)$ is in phase with $B(t)$ where the peak value is the same in both cases.

Thus:

$$\Delta IC''' = 20 \log_{10} \frac{1 - M_1(1 - K)}{1 - M_1(1 + K)}$$

$$RIC''' = 20 \log_{10}(K_1 + K_2) + 20 \log_{10}[1 - M_1(1 - K)]$$

Note that $\Delta IC'''$ and RIC''' now depend on ΔIA, i.e., $M_1 = f(\Delta IA)$

since $\Delta IA = 20 \log_{10}(1/1 - 2M_1)$

Defining $x = IA/20$ and $y = 10^x$, we obtain IC''' and RIC''':

$$\Delta IC''' = 20 \log_{10} \frac{1 - .5(1 - 1/y)(1 - K)}{1 - .5(1 - 1/y)(1 + K)}$$

$$RIC''' = 20 \log_{10}(K_1 + K_2) + 20 \log_{10}[1 - .5(1 - 1/y)(1 - K)]$$

Case III: This section will be concerned with the possibilities of determining parameters for the model shown in Figure 3. To do this, a dual channel MAID audiometer with independent controls for each channel will be needed. The interaction and properties of the various neural stations (represented by symbols T_d, K_1, and F) are at present only partially defined and it will be a task of this study to empirically determine their basic roles in hearing.

Defining the stimuli at inputs A and B as:

$$A(t) = G_1[1 + M_A(t)] \sin (W_c t)$$

$$B(t) = G_2[1 + M_B(t)] \sin (W_c t)$$

where $M_B(t) = G_3 M_A(t + b)$ where $0<b<T/2$

The result will be stimuli at A and B that are equal in carrier frequency but variable in intensity, ΔI, and phase. However, all amplitude changes in the carrier frequency will still be in synchronization with its zero crossing.

The T_d for input A is incorporated in the model to allow for possible time delays in the block described by $C''' = K_1 A + K_2 B$. Interaural action is such that equal bilateral stimulation may not be perceived as such. Therefore, given Cases I and II (i.e., A = B, A = B but 180° out of phase regarding modulation signal), a slight shift of phase (for example, 5° or 185°) could possibly create an entirely different perceived phenomenon. Conversely, in the case of central neural involvement, the phase relationship could be perceived as being contrary to Cases I and II when, in fact, it is not. The block represented by $\Delta IA = F(SL)$ is to account for the "true" ΔI perceived at C as a monotonically decreasing function of sensation level (SL), where SL = [stimulus intensity minus hearing threshold level (HTL)].

Summary of Proposed Central Testing Procedure and Apparatus

The rationale for this proposed research is based on neural integration which involves a "summing effect" of independent stimuli in each ear at the brain level. For example, if 60 dB of noise is independently introduced into each ear, the integration effect would cause a subject to perceive a noise of approximately 63 dB. Up to the present, the major efforts in central testing have used speech as the stimulus of choice. The nature of the MAID stimulus is such that it appears to be well suited to the integration tasks since it can be manipulated independently at each ear with predictable single perceptual correlates. Stated another way, it appears that no two sets of stimulus parameters at ear A and ear B will yield the same perceived stimulus to a subject. At the same time, the subject need make only one judgement rather than trying to "pick out" meaning (as in speech). The preceding mathematical model is intended to show the wide variety of stimulus parameters possible using the MAID device along with formulated predictions of perception, depending on the hearing loss. Once case, simply stated, is that the ΔI stimulus can be

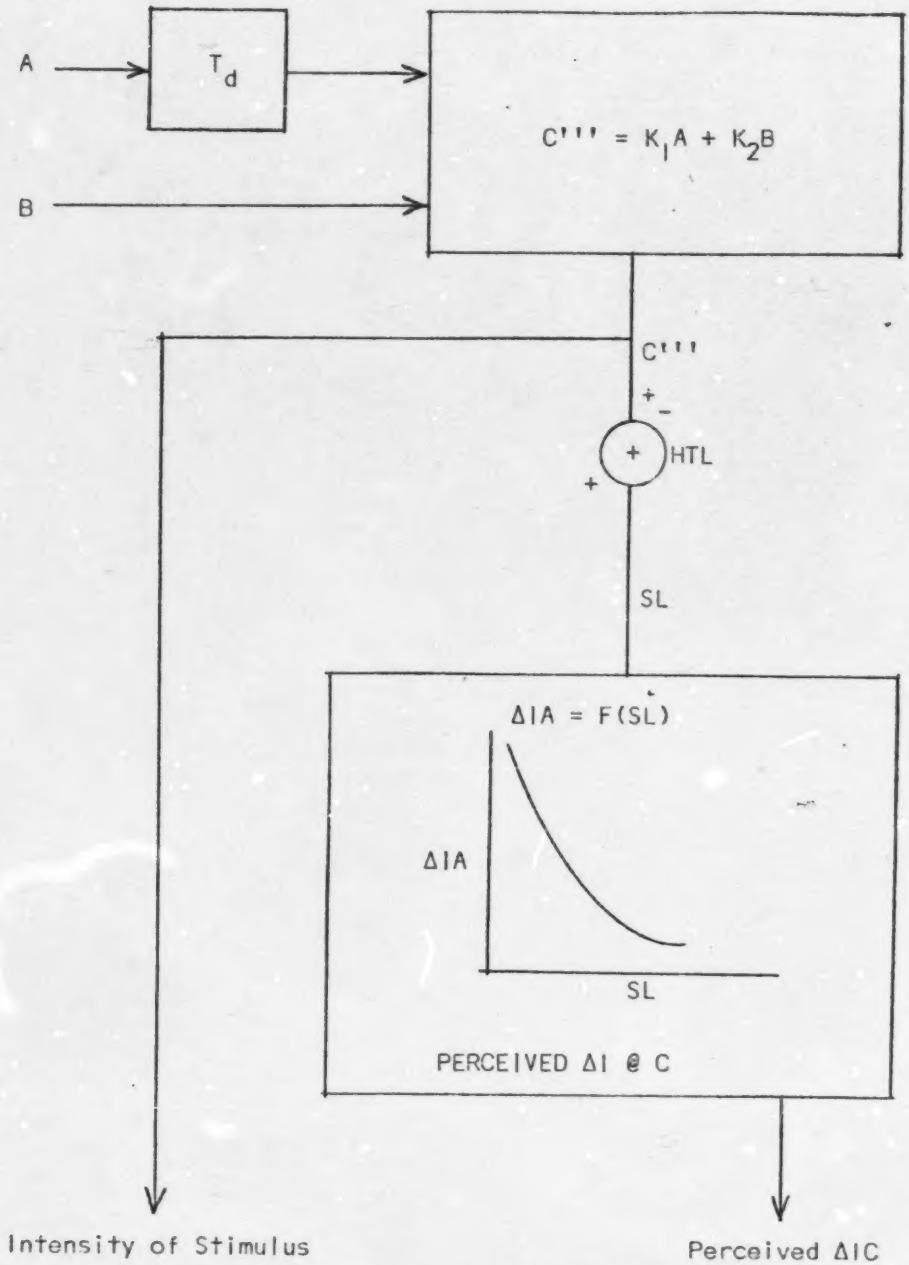
A
T_d
B
$C''' = K_1A + K_2B$
C'''
+
−
+
HTL
+
SL
ΔIA = F(SL)
ΔIA
SL
PERCEIVED ΔI @ C
Intensity of Stimulus
Perceived ΔIC

Figure 3

presented in ear A and ear B 180° out of phase and, as a result, integration should "smooth out" the changing function. Figure 4 illustrates this point.

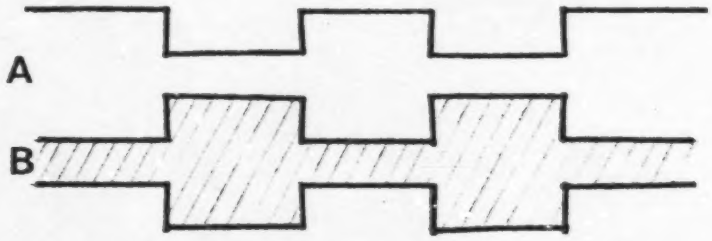

Figure 4

Notice that the meshing of the stimuli reduces the ΔI perception. Another possibility will be the allowance of neural lag by simply letting the subject match up the A and B stimulus to equality. See Figure 5.

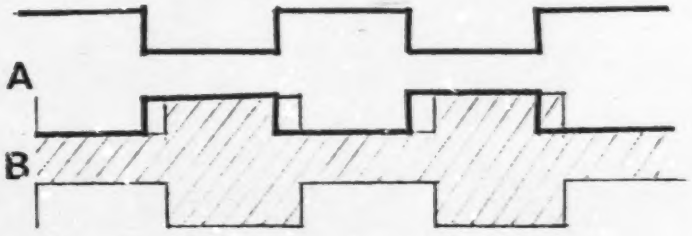

Figure 5

Using these techniques, it is felt that all findings from procedures using speech can be duplicated, plus more definite statements about site of lesion.

Tables I through 5 are based on Figure 6 which is a schematic of the hearing neural apparatus beginning at the cochlea and ending at the cerebral cortex. Each neural station is assigned a loss (i.e., KA = 0 means no transmission at that point) and the effects of the stimuli on C''' is then shown for all conditions.

Example: assume a total loss at KA (KA = 0), resulting in only stimulus B reaching the cortex. Referring to Table I, if KA = 0 then the stimulus at C''' = .2A + .8B and all characteristics are equal to the input stimulus A. If stimulus A is 180° out of phase (see Figure 4 with stimulus B, then referring to Table 2 it can be seen that C''' = .2A + .8B but the other parameters of C''' change according to the ΔI input.

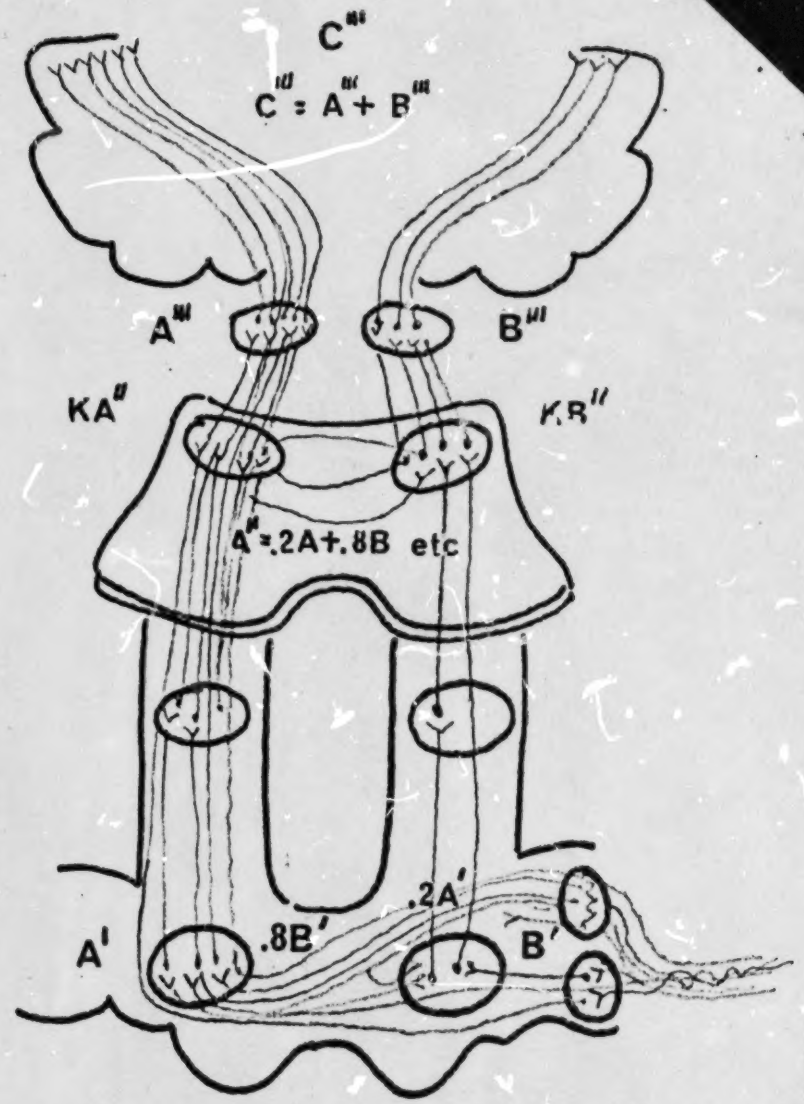

Figure 6

TABLE I

$A(t) = B(t)$

Loss	Formula for C'''	RIC''' in dB (Peak Intensity Change)	ΔIC in dB	Phase of C'''
KA = 0	C''' = .2A + .8B	0	Equal to Input at A	Equal to Input at A
KB = 0	C''' = .8A + .2B	0	Equal to Input at A	Equal to Input at A
.8A' = 0	C''' = .2A + B	1.58	Equal to Input at A	Equal to Input at A
.8B' = 0	C''' = A + .2B	1.58	Equal to Input at A	Equal to Input at A
KA'' = 0	C''' = .8A + .2B	0	Equal to Input at A	Equal to Input at A
KB'' = 0	C''' = .2A + .8B	0	Equal to Input at A	Equal to Input at A
.2A' = 0	C''' = .8A + B	5.10	Equal to Input at A	Equal to Input at A
.2B' = 0	C''' = A + .8B	5.10	Equal to Input at A	Equal to Input at A
No Loss	C''' = A + B	6.0	Equal to Input at A	Equal to Input at A

TABLE 2

A(t) = B(t + T), i.e. B(t) 180° out-of-phase with A(t)

Loss	Formula for C'''	(Peak Intensity Change)	ΔIC''' in dB	Phase of C'''
KA = 0	C''' = .2A + .8B	Table 3	Table 3	Equal to input at B
KB = 0	C''' = .8A + .2B	Table 3	Table 3	Equal to input at A
.8A' = 0	C''' = .2A + B	Table 4	Table 4	Equal to input at B
.8B' = 0	C''' = A + .2B	Table 4	Table 4	Equal to input at A
KA'' = 0	C''' = .8A + .2B	Table 3	Table 3	Equal to input at A
KB'' = 0	C''' = .2A + .8B	Table 3	Table 3	Equal to input at B
.2A' = 0	C''' = .8A + B	Table 5	Table 5	Equal to input at B
.2B' = 0	C''' = A + .8B	Table 5	Table 5	Equal to input at A
No Loss	C''' = A + B	See Note 1	See Note 1	See Note 1

Note 1: Not applicable because C'''(t) = 0.

TABLE 3

Losses: KA = 0, KB = 0, KA'' = 0, KB'' = 0

ΔI at C''' in dB	ΔI at A in dB
7.248E-6	0
.299929	.5
.599557	1.0
.898556	1.5
1.19661	2.0
1.49338	2.5
1.78859	3.0
2.08192	3.5
2.37308	4.0
2.66177	4.5
2.94773	5.0
3.23067	5.5
3.51033	6.0
3.78643	6.5
4.05875	7.0
4.32706	7.5
4.59111	8.0
4.85071	8.5
5.10567	9.0
5.35582	9.5
5.60098	10.0

RIC''' in dB	ΔI at A in dB
-3.83108E-5	0
-9.77941E-2	.5
- .191065	1.0
- .280046	1.5
- .364897	2.0
- .445764	2.5
- .522837	3.0
- .596199	3.5
- .666049	4.0
- .732503	4.5
- .795706	5.0
- .855814	5.5
- .912924	6.0
- .967189	6.5
-1.01874	7.0
-1.06769	7.5
-1.11415	8.0
-1.15823	8.5
-1.20007	9.0
-1.23975	9.5
-1.27737	10.0

TABLE 4

Losses: .8A' = 0, .8B' = 0

ΔI at C''' in dB	ΔI at A in dB
7.248E-6	0
.333262	.5
.666239	1.0
.998607	1.5
1.33005	2.0
1.66028	2.5
1.98897	3.0
2.31585	3.5
2.64062	4.0
2.96301	4.5
3.28271	5.0
3.59946	5.5
3.913	6.0
4.22304	6.5
4.52934	7.0
4.83166	7.5
5.12975	8.0
5.42344	8.5
5.71247	9.0
5.99661	9.5
6.27576	10.0

RIC''' in dB	ΔI at A in dB
1.58362	0
1.50223	.5
1.42472	1.0
1.3509	1.5
1.28063	2.0
1.21377	2.5
1.15016	3.0
1.0897	3.5
1.03221	4.0
.977584	4.5
.925713	5.0
.876451	5.5
.829696	6.0
.785313	6.5
.743212	7.0
.703261	7.5
.665402	8.0
.629485	8.5
.595461	9.0
.563197	9.5
.53264	10.0

TABLE 5

Losses: .2A' = 0, .2B' = 0

ΔI at C''' in dB	ΔI at A in dB
7.248E-6	0
5.55435E-2	.5
.110984	1.0
.166251	1.5
.221241	2.0
.275878	2.5
.330077	3.0
.383758	3.5
.436825	4.0
.489218	4.5
.540861	5.0
.591688	5.5
.641637	6.0
.69065	6.5
.738673	7.0
.785669	7.5
.83158	8.0
.876385	8.5
.920043	9.0
.962533	9.5
1.00383	10.0

RIC''' in dB	ΔI at A in dB
5.10536	0
4.88665	.5
4.67504	1.0
4.47041	1.5
4.2727	2.0
4.08183	2.5
3.8977	3.0
3.72023	3.5
3.5493	4.0
3.38478	4.5
3.22652	5.0
3.07444	5.5
2.92838	6.0
2.78819	6.5
2.65374	7.0
2.52488	7.5
2.40144	8.0
2.28327	8.5
2.17022	9.0
2.06212	9.5
1.95882	10.0

Statement Of The Problem

A specific audiometric finding in LD's has been mentioned earlier in this paper. This section describes those findings.

"Normal" audiometric findings vary depending on other factors. For school screening purposes, "normal" usually means an air conduction screening at 250, 500, 1000, 2000, 4000, and 8000 Hz at 20 dB. However, to an otologist treating otitus media, this level of screening would not pick up the information needed. Also, "normal" is often related to school progress. That is to say, a child with a mild loss is considered to have "normal" hearing if all other activities are unaffected. On the other hand, a child with a mild loss who is failing in school might be considered hearing impaired to a degree to warrant the use of a hearing aid.

A mild hearing loss is defined here as part or all of the pure tone threshold lying between 30 dB and 45 dB Re HTL. If discrimination is involved, the definition changes but usually no such involvement is found in a mild loss except where that loss is of a central nature. Thus, if a child is having learning problems and any portion of the audiogram shows an unexplained loss, that loss becomes significant and not within normal limits. Such a loss is the basis of this proposal.

In the routine practice of audiology, the audiogram shown in Figure 7 has often been encountered by this writer in children who have been labeled LD.

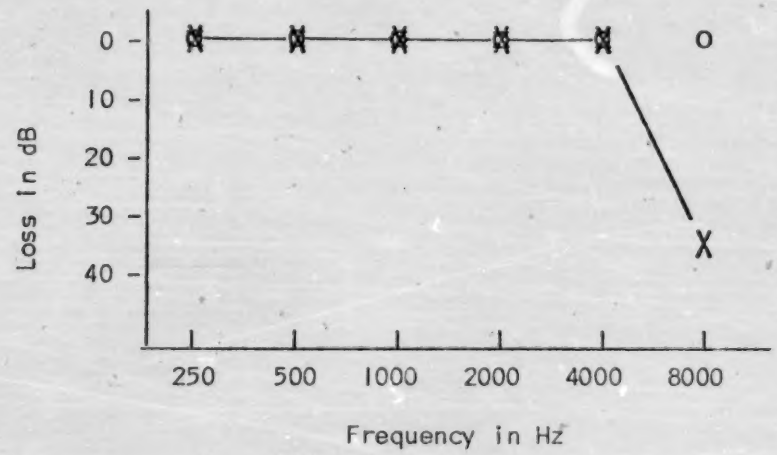

Figure 7

No qualifications of this pattern exists beyond discussion with others in the field of hearing diagnosis who have observed the same pattern. The configuration of the loss is suggestive of a specific neural lesion of some type, but beyond this, no cause can be stated. Therefore, no

lengthy discussion is warranted since the observation stands alone, unquantified, unqualified, and existing in cases where the label LD might be arbitrary. It is the purpose of this study to test labeled LD children using the Minimal Auditory Intensity Differential which was discussed in detail above.

Subjects

Subjects for this study will be drawn from school systems in the immediate vicinity of New Mexico State University. There will be an experimental group of children drawn only from LD classes and a control group equal in number to those labeled LD drawn randomly from regular classrooms. Efforts will be made to control all variables such as age, language, social and educational background, sex, and techniques for previous evaluation and placement.

Procedure

Each subject will be carried through a sequencing including:

1. Pure tone thresholds at 500, 1000, 2000, 4000, and 8000 Hz.
2. Unilateral MAID scores.
3. Binaural MAID scores.
4. MAID Lead or Lag.

The first observable data should be simply the presence or absence of specific loss, particularly at 8000 Hz unilaterally. Standard statistical analysis will be carried out to specify differences between the experimental and control groups.

Pilot Data

Thirty-six subjects were tested in a pilot study that utilized the same subjects as McKenney (1976) in her analysis of sound blending skills in reading and articulation impaired children. The subjects were divided into three groups of 12 each -- one group with normal reading and speech skills, one group with below grade-level reading skills and normal speech skills; and one group with grade-level reading skills but exhibiting an articulation problem characterized by one or more defective speech sounds.

The testing procedure previously described was administered to each subject. Figure 8 presents a plot of data for the three groups based on a count of frequencies that deviated from the responses of normal adults.

As Figure 8 indicates, there appears to be a trend in the articulation defective children away from the groups composed of reading defective and normal children. This trend parallels the difference found by McKenny for the articulation group. McKenney's finding of a difference between the normal and the reading group did not appear.

The purpose of this study will be to further investigate the differences between the three types of children in a much larger population.

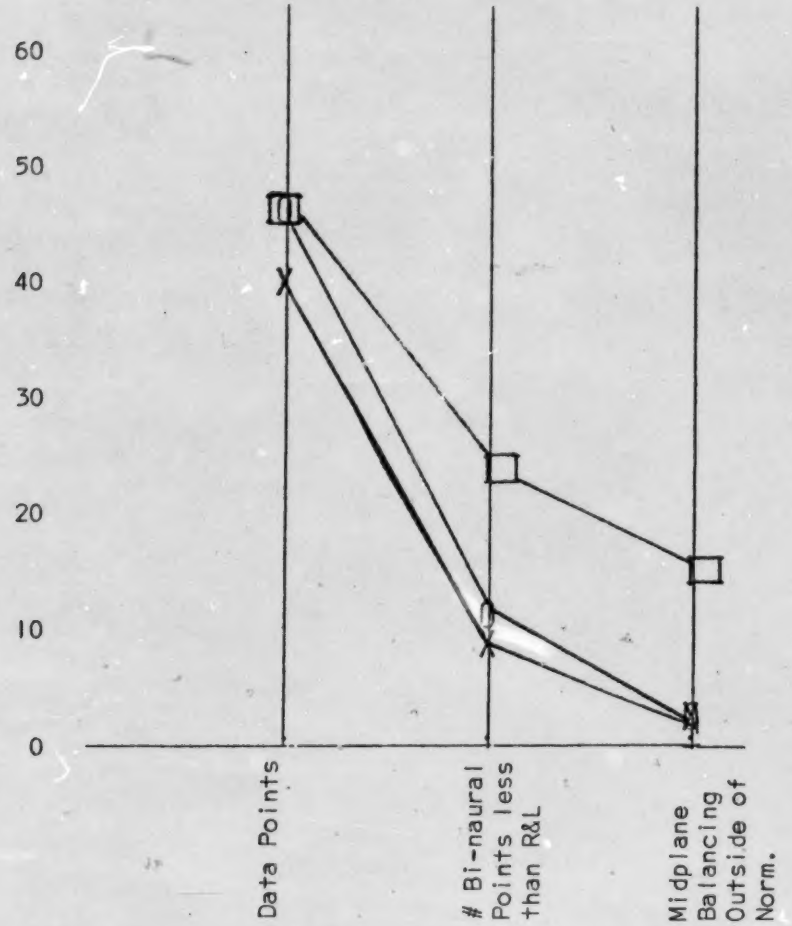

Figure 8

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